Supplementary Table 1. *A list of foundational and recent studies utilizing patient-derived/human iPSC-CMs to uncover the functional role of cardiac ion channels and dyad proteins in eliciting arrhythmogenesis.*

	Condition	Study	Gene Variant	Characteristics of mutant human iPSC-CMs compared to healthy controls
Foundational studies on specific gene variant-induced iPSC-CM-based arrhythmia models	BrS	(Kosmidis et al., 2016)	SCN5A p.(Arg1638X)	1. Significantly reduced V_{max} 2. Reduced peak I_{Na} density 3. Reduced cardiac AP upstroke velocity
	CPVT	(Fatima et al., 2011)	RYR2 p.(Phe2483Ile)	1. Increased DADs and arrhythmias when exposed to adrenergic agonists 2. Higher amplitudes and longer durations of spontaneous local Ca^{2+} release events
	LQTS	(Moretti et al., 2010)	KCNQ1 p.(Arg190Gln)	1. Prolonged APD 2. Reduction of I_{Ks} 3. Enhanced vulnerability to catecholamine-induced tachyarrhythmia, which is reduced with beta blockers
	SQTS	(El-Battrawy et al., 2018)	KCNH2 p.(Asn588Lys)	1. Shortened APD 2. Increased I_{Kr} and hERG
Expansion and optimization of gene-variant induced iPSC-CM-based arrhythmia models		(Selga et al., 2018)	SCN5A p.(Arg367His)	1. Reduction in I_{Na} peak current density 2. Accelerated recovery from Na channel inactivation 4. Similar changes I_{Na} properties observed across different iPSC-CM differentiation procedures
		(de la Roche et al., 2019)	SCN5A p.(Ala735Val)	1. Reduced cardiac AP upstroke velocity due to reduced I_{Na} inward 2. Right shift of Na activation curve, prolonged recovery from inactivation
	BrS	(Li et al., 2020)	SCN5A p.(Ser1812X)	1. Reduced peak I_{Na} density 2. Irregular Nav1.5 localization and decreased colocalization of Nav1.5 and Cx43 3. Reduced Nav1.5 expression 4. Reduced cardiac AP upstroke velocity

				5. Abnormal AP profile, including EADs and DADs 6. First demonstration of conduction slowing in BrS-CMs
		(Lu et al., 2023)	SCN5A p.(Arg620His)+ p.(Arg811His)	1. I_{Na} density reduced, lower NaV1.5 2. Beating interval variation
	CPVT	(Itzhaki et al., 2012)	RYR2 p.(Met4109Arg)	1. No observed difference in AP properties 2. Increased occurrence of DADs 3. Significant whole-cell Ca^{2+} transient abnormalities, which aggravated upon adrenergic stimulation but improved with β-blockers
		(Novak et al., 2012)	CASQ2 p.(Asp307His)	1. Prolonged APD 2. DADs and diastolic Ca^{2+} increase caused by β-adrenergic stimulation
		(Zhang et al., 2013)	RYR2 p.(Phe2483Ileu)	1. Caffeine-induced Ca^{2+} transients produced smaller I_{NCX}, showing smaller Ca^{2+} stores 2. Higher CICR gain 3. Increased diastolic Ca^{2+} level and spontaneous Ca^{2+} release
		(Acimovic et al., 2018)	RYR2 p.(Asp3638Ala)	1. Abnormal Ca^{2+} release under stress 2. Decreased Ca^{2+} transient amplitude under stress 3. Increase in systolic and diastolic aberrant Ca^{2+} events under stress 4. Leaky RYR2 channels under stress
	LQTS	(Itzhaki et al., 2011)	KCNH2 p.(Ala614Val)	1. Prolonged APD 2. Reduction of I_{kr} peak amplitudes at depolarization steps 3. Development of EADs
		(Egashira et al., 2012)	KCNQ1 1893delC	1. Prolonged Field potential duration (FPD) 2. Treatment of LQTS-iPSC-CMs with isoproterenol induces ventricular tachycardia-

				like arrhythmia, which was blocked by β-blocker
		(Ma et al., 2013, p. 3)	SCN5A p.(Val1763Met)	1. Increased late I_{Na} and APD prolongation 2. Shortened I_{Na} inactivation recovery
		(Malan et al., 2016)	SCN5A p.(Arg1644His)	1. Prolonged APD 2. High incidence of EADs 3. Accelerated recovery from Na current inactivation
		(Garg et al., 2018b)	KCNH2 p.(Thr983Ile)	1. I_{kr} density reduced 2. High diastolic Ca^{2+}